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Clinical Proceedings

CHILDREN'S HOSPITAL

OF THE DISTRICT OF COLUMBIA

What to Tell the Parents of a Child with Cancer
Reginald S. Lourie, M.D., James Hatleberg, M.D., Grace H. Guin, M.D. and Sanford L. Leikin, M.D. 91

Congenital Glaucoma
Frank D. Costenbader, M.D. and Marvin L. Kwitko, M.D. 100

Chronic Lead Intoxication: A Report of 24 Treated Cases at Children's Hospital
Nicholas P. Haritos, M.D. 110

The Editor's Column: Pediatric Ophthalmology Needs a Children's Hospital 115

Book Reviews 118

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Clinical Proceedings

CHILDREN'S HOSPITAL WASHINGTON, D.C.

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Panel Discussion: What to Tell the Parents of a Child with Cancer

Reginald S. Lourie, M.D.*
James Hatleberg, M.D.†
Grace H. Guin, M.D.‡
Sanford L. Leikin, M.D.§

Dr. Lourie:

The task of what to tell parents of a child who has cancer is about as difficult as anything one comes in contact with in the entire practice of pediatrics. As we are better able to handle the acute infectious diseases we will be seeing more chronic diseases, so that the handling of terminal illnesses becomes a very practical matter. This aspect of the problem—the handling of the parents as well as the child—falls naturally in the domain of the pediatrician, and rightfully so.

Dr. Hatleberg will briefly summarize the courses of two children with terminal illness whom we have observed recently in this hospital with particular reference to how the children, their families, and house staff reacted to this situation. Dr. Guin and Dr. Leikin, who have a wide clinical experience in the handling of this type of situation, can comment on the clinician's point of view, and finally, I will try to summarize from the psychiatric viewpoint.

Dr. Hatleberg:

Case 1

This 4½ year old girl was brought to the hospital because she had fallen from a tree and complained of abdominal pain. A very alert physician recognized a tumor

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in the abdomen and the child was hospitalized. Following diagnostic study, the child went to surgery where a metastatic tumor was discovered. The mother was quite concerned and felt guilty because she had let the child climb the tree; she expressed this to the doctor in charge of the case. He attempted to reassure her and tell her that the tumor was in no way the result of falling out of the tree, and in his subsequent contact with her he tried to give her some understanding of the tumor. However, he apparently had difficulty in talking to her about it, and so, probably sensing that she did have guilt feelings, told her that while this was a tumor which was fatal in many children, he still could not say that it would be fatal to her child. At the time he said this it was already known that the tumor had widely metastasized.

The child's course on the ward following surgery was one of difficulty in management for the ward personnel. There was apparently a change in the child's behavior; she was having frequent temper tantrums, and was crying, screaming, and kicking her feet a great deal. In her play on the ward she had a doll which was given to her, and after recovering from surgery she would take the doll and hit it in the abdomen as hard as she could and say it was a bad doll and had to be spanked. After she had become less violent in her play with the doll, she still thought that the doll was "the bad doll" and she would finger it and show an exaggerated amount of interest in its abdomen. In making rounds, the doctor in charge of the case had increasing difficulty in stopping by the bed of this child. As the child began to lose physical strength, his visits to the bedside became briefer and briefer. In his own mind, he was always wondering whether there was not some other reason why the child did not have the strength she used to have. The little girl became less interested in people on the ward, wanted to stay in bed more, did not want to watch television, and finally became lethargic and comatose.

The parents did not visit the child very frequently during the hospitalization. They had been told that this might be a serious illness, and their concern for the child was such that it was difficult for them to visit. They moved out of their apartment, gave up the care of their two other children to the maternal grandmother, and lived isolated in the country. Toward the end of her illness, her father, to whom this child was very important (his favorite child), had difficulty in coming to visit and always had to drink alcohol before he could come. He would walk into the room and say, "Hello, honey," talk to her a little bit, and then, as his eyes started to tear up, would walk out of the room. The mother, who had a lot of feeling for the child too, had even more difficulty, but expressed it in a different way. She had had several spontaneous abortions and felt badly about them, but in coming to see the child, she was trying to convince herself that the child was not really sick. She would ask in a suspicious way what the doctors were doing with her child with all those tube feedings and intravenous needles they were giving her which were hurting her. "Why are they doing that?"

As the child's illness became terminal and it was obvious that she was not going to live very long, the nursing office on the ward notified the parents, who came to the hospital. They did not see the doctor at this time, and they still did not have the idea that the child was more than seriously ill. Finally, it was impressed upon them that the child was going to die and that it would be wise to be available not only for the child but for their own feelings as well. When the child did die, the mother later told one of the social workers in the hospital, "If only the doctor had told us that she was going to die."

Case 2

This 14 year old child had a spina bifida and was operated on in infancy; an osteomyelitis resulted. During her entire lifetime the osteomyelitis was uncontrollable

by any antibiotic medication and the disease destroyed so much of the pelvis that she was paralyzed from the waist down. She had a long course of illness during which she had to be taken care of as a bed patient by her mother. At the time of her last hospitalization, it was realized that this was a terminal case and the parents were prepared. The child herself was a smiling pleasant girl who appeared to be very friendly, but this was thought to be a surface manifestation of rather serious depression. As time went by and the child wanted to go home but could not, she would become depressed to the point where intravenous fluids and blood transfusion had to be employed to maintain life.

The doctor taking care of the child then began sitting down after he started the intravenous fluid and spending about half an hour just talking to her. The results of his therapy were rather obvious because, following his visits, she would always start to eat again and would again be interested in the people around her and in what was going on on the ward. But as time passed and the illness progressed, she became lethargic and lost interest in people whom she had known on the ward and with whom she had been friendly. She started having seizures, and when asked about this, the response was, "I don't want to know about it." During the final phases of her illness, her mother and father made arrangements to be with her as much as possible. They recognized that this was really the end of her illness and that she was going to die. They could comfortably be with her and were very appreciative of what the hospital had done for her.

Dr. Guin:

When a physician's practice includes a number of patients suffering from leukemia and malignant solid tumors, he is afforded an excellent opportunity for developing a close personal relationship with both patient and parent. The establishment of such a bond of feeling has been, in our experience, a source of strength for the distressed parents. During the time the physician is treating the child he may become counselor to the family. This relationship is not only important from the emotional standpoint of parents, but serves to insure that the patient will receive standard accepted treatment. I am referring to a situation that frequently arises in a household once a diagnosis of cancer has been made. The distraught parents may be besieged by well meaning neighbors, members of various sects, and quacks, each sponsoring his own brand of magic cure or plan of management. The parent, being in a desperate state of mind and not wanting to overlook anything that might help, is buffeted about on the sea of confusion, thus adding to his already heavy burden. It is the role of the physician to listen to the parent, explain the futility of following blind leads, and briefly bring him up to date on what acceptable therapy includes. Such patience and understanding on the part of the physician will make for close cooperation, and help, in time, ease some of the burden as the parent comes to accept the fact that his child is receiving the best treatment available.

Under circumstances where the course of the disease is less protracted, the physician does not have as long a period to become acquainted with the

family, but he can certainly be helpful during those times too. There are problems which arise when the patient does go home after having received a course of x-ray treatment for a neuroblastoma or a brain tumor, for example. The parents may ask, "What will happen? What is likely to happen between today and the next time we bring the child in? Will he be seized with excruciating episodes of pain? Will he go blind? Will he die suddenly? Will he become paralyzed? Will he hemorrhage?" All of these things are frightening because they are possible. Again, the physician can help immeasurably by informing these parents of what they need to know about the possible course and the future of the disease; it is important not to emphasize the worst aspects. They should feel that the physician is available if they do meet a situation which they feel unable to handle.

What do we say when asked about the possible duration of the illness? This, of course, is highly speculative and we attempt never to go out on any limb in answering this question. The only figures we could quote would be that in the group of acute leukemias, 75 per cent of the patients are dead at the end of six months, but we emphasize that we cannot tell which child falls in the 75 per cent category and which child falls in the 25 per cent category. Since we do not know after starting treatment on a patient with leukemia whether he will live seven days or seven years, it seems unfair to give the parents a definite interval of time, such as six months, a year, or two years, because they fix that in their minds and are unhappy over knowing that such a date is certainly approaching. Employing chemotherapeutic agents makes the future even more uncertain. We have some children with neuroblastomas, for example, who have survived over five years, and although we explain that the tumor is practically always fatal, there have been rare cures or regressions.

What positive suggestions do we have that might be helpful to the parents? When the parents are young we have suggested that they think about having another child, pointing out that the disease is not inherited and that the likelihood of other children having the disease is rare. One thing I find helpful in talking to the parents is stressing the fact that they should not neglect the other siblings because this particular child has been stricken.

I do not think the physician should try to force his religion on the patient or attempt to force the parents to tell the child that he is going to die. If the parents want to tell the child, this is up to them. We have never felt that we would ever tell a child he is going to die.

Almost without exception, the parents of these children react remarkably well when they are told that their child has a fatal disease. I have had only one case in which the father of a patient with leukemia refused to let me go on with treatment because he said that the continued illness of the

child was upsetting his wife and he would have to commit her unless the child died as quickly as possible. There was no choice other than withdrawing treatment, and the child died shortly thereafter.

I feel that the parents should be forewarned when the child is admitted for what appears to be the last time. I find it helpful at this time also to explain to the parents in language that they can understand a little bit about the physiopathology. They see tubes attached to the arms and inserted in the stomach, pills being taken, blood being withdrawn, and they feel better satisfied if someone explains something about the treatment at this point.

In the majority of cases both parents seem to share the emotional load equally. I think the feelings of parents who have lost a child from a tumor are somewhat different from the feelings of the parents who have lost a child from a sudden acute illness. The parents of a child who has died from a tumor have already adjusted to the fact that the patient had an unusual type of illness from which he would most certainly not recover. They have been tormented by the suffering that the child has undergone during the final stages of his illness and they have been exhausted by the frequent visits to the hospital and sitting by the bedside for long periods of time. They have been forced to neglect, to a certain extent, the siblings and the care and management of their home. Some parents look upon the end as a relief to the child from his suffering. What can the physician say to parents at this particular time? I think it is important for the parents to feel that the child did not really suffer as much as he seemed to. It is important, therefore, for the physician to make a point of mentioning this whether it is true or not. One night, a father called me a month following his child's death and he said, "Do you think that he suffered very much that last day?" If he had just mentioned it or if I had thought to tell him that last day, perhaps he would not have been entertaining this uncomfortable thought for such a long period of time. Of course, it is assumed that the physician has seen to it that the patient had been made as comfortable as possible so that he would not have suffered any more than was necessary.

How does the child react to certain restrictions inherent in the disease process as the end approaches? Children with brain tumors or tumor metastases to the brain, for example, not infrequently become blind just prior to the end. Blindness in many instances is accepted without any panic on their part. They sit in the television room and are content to listen to the program, rarely referring to their blindness and not seeming depressed over it in any way. A paralyzed child may accept being pushed up and down the hall in a wheel chair without any comment pertinent to the situation.

One of our 6 year old leukemia patients was informed by his playmate, Willie, that he would die from his disease. Our patient, somewhat panic

stricken, ran into his house and asked his mother it it was true that he was going to die. His mother, a very intelligent and resourceful woman, replied after catching her breath, "Of course, you are going to die, so am I, so is Daddy, and so is little Willie." This logical retort seemed to put Willie's announcement it its proper frame of reference, and our patient gave it no further thought.

With the older child, the problem of how to handle the question of death becomes a little more complex. We recently had a 13 year old boy with a lymphosarcoma who had a frozen pelvis and a functioning colostomy. He had been told that he had a draining abscess from a ruptured appendix. As far as we could tell, we felt that he accepted this diagnosis as correct. We never intended for him to know otherwise. Another boy, 15 years of age, who had leukemia (in remission), was being discharged from the hospital and somehow found out what his diagnosis implied. He solved his problem by leaping from his eighth story hospital room. Whether his action was triggered by the cortisone he was receiving, we will never know.

There obviously are many facets to the problem of how the physician should handle the question of what to tell the parents of children with cancer. There is no single answer to this question. In addition to medical knowledge, it is important to mention again that the physician should have a feeling of closeness with the patients and a keen insight into the situation in which the parents find themselves.

Dr. Leikin:

My remarks will be in reference to leukemia and those hematologic diseases with fatal prognoses. They amount to a series of do's and don't's based on experience and, to a certain extent, on my own personal philosophy.

To begin with, I feel that even before the consultant actually sees the patient, the family should be made aware of why the consultant is being asked to see the child. The seed, so to speak, is thus planted, and when the diagnosis is established, the shock is not as unexpected or as overwhelming as it might otherwise be. I do not mean to imply that definitive terms like leukemia should be used in preparing the parents for such an eventuality; this should be best left for the consultant after he is sure of what the diagnosis actually is. Once the diagnosis is established, a full, clear, and concise explanation should be given. Lay terms and explanations should be the rule, and although the physician should not set himself up as infallible, he should not leave any doubt in the parents' minds. Doubt can only lead to suspicion and fear. The parents may then spend endless hours of shopping around looking for what they want to hear. In the end, this makes the despair and heartache only greater. Should it be necessary (in

terms of the parents' acceptance) then let the physician be the one to suggest an additional consultation to corroborate the diagnosis. Such a practice is most desirable.

Assuming that the diagnosis has been accepted, the idea of eventual death is not usually as readily accepted. One soon sees the results of guilt feelings in the parents. "Maybe I should have called the doctor sooner." "If I had only been more careful and Johnny hadn't fallen. . . ." "Is this disease something the child inherited from one of us?" Answers and reassurances to all of these and many more questions must be given and repeated in a gentle and understanding way.

Inasmuch as treatment at best is palliative and not curative in leukemia, the decision as to whether or not to institute treatment must rest with the parents. However, we always point out that even though we cannot talk in terms of a cure for the child, we can certainly offer a prolongation of life. Making the child comfortable is even more important. It is equally useful to point out to the parents that once therapy is begun and even a remission achieved, it is only that, and the eventual outcome will not be really any different from what it was prior to treatment. The parents should be told of what the treatment consists, and that the treatment is fairly well standardized throughout the various centers treating children with this disease. This will tend to alleviate the endless search for a place or a man who might offer something more or different. When the child enters the terminal phase of his disease, it becomes increasingly important for the pediatrician in charge of the case to be even more tolerant and understanding than usual. The parents may request various things be tried and added to the child's treatment. Even though the physician normally does not permit parents to recommend specific therapy for his patient, I believe under such conditions the physician would do well to accede to such requests. Thus, afterward, the parents will not say, "Maybe if we had tried. . . ." Naturally, the requests have to be reasonable; nor should the physician give up his role as the one who is in charge. We do not tell the child that he is going to die. We advise the parents not to neglect the other children in the family.

Dr. Lourie:

We are indebted very much to Dr. Julius Richmond who has done probably more definitive work than anyone else in studying this phase of illness in a pediatric setting. We can understand the parent reaction to the death of the child in psychological terms as a form of separation anxiety. Separation anxiety is a normal phenomenon which comes up very early in a child's life. Separation is a problem which creates anxiety at every level of development where there is a break from the parent, for example, in developing independence. The nature of the anxiety which the parents have

in the face of a terminal illness is basically that of separation anxiety, but its impact and meaning are quite different in a situation of impending death: this time there will be a permanent separation.

In the terminal phase of chronic illness in a child the normal process of mourning by the parents which we expect takes place before the child dies. It is very important to keep this in mind, and to keep in mind also the problem of allowing these parents to have this period of mourning. This period of mourning will involve certain usual processes; it will involve, first of all, a removal of interest and energies from everything else, and investing these back into themselves. It involves a process of self-examination and self-condemnation; guilt arises in many different ways. We have to realize that this is a normal part of the mourning process. Not only do we have to allow a parent to go through this process, but we have to give them permission to do so. We also have to make it possible for some of this guilt to be worked out. As we have heard from Dr. Guin and Dr. Leikin, the essence of being able to do this is in the relationship of the doctor with the parent. In other words, one has to be available and one has to be realistic. Somebody has to present the situation as it is. Once we give parents the opportunity to bring up what they feel guilty about, the most amazing little details come up. They talk about such things as, "If I only hadn't made him go to school every morning," or, "If I didn't stop him from watching television and let him get mad, then this would not have happened." We can then be in a position to deal with these feelings of guilt.

In the process of recovering from the mourning, the next step is a reinvestment of the energies that had been withdrawn. Here too, the doctor is in a strategic position to help the redirection of the energies that have again become available. As we have heard, these energies can be made available to deal with the child, to help the child, and to work with the child. They also can be redirected back to the rest of the family. As we watch parents during these terminal phases we note a very interesting phenomenon: when their guilt has been worked out, they develop interest in other children, interest in helping out on the ward if they can be allowed to, and interest in the disease itself. It is the parents of children who have been through these fatal illnesses who have been the moving factor in large foundations dedicated toward the study of cancer, leukemia, cystic fibrosis and other chronic diseases that end fatally. How much good has emerged from the way in which we, as doctors, have been able to direct some of these energies that have become available again into useful channels!

The child's reaction can be very puzzling to us. Why is this child not upset about the fact that he is going to die? There may be exceptions where it has not been presented properly, or the child is an older child, or has

some special problem around death or in his picture of himself. The problem in the child centers around the loss of energy. The less energy available, the less the child has to invest in the surroundings.

Another principle that we have to keep in mind is the child's idea of what death is. The younger child particularly does not think of death as something permanent. He thinks of death as being temporary and even normal. Recently a three year old did not want his parents to go out for the evening. He said, "I hope you die. I hope you get hit by a car, but when you come back, bring me a lollipop." In other words, death is associated in a child's mind with something reversible but always concerned with violence. Since violence is not involved in the terminal illness, the child is not afraid of death in terms of the way he sees death. He is, however, afraid of death from his illness as punishment. In the first case that Dr. Hatleberg presented, the child was blaming herself for being sick, for having symptoms. Here again, when we become aware of such feelings, we need to help the child with them by reassuring him that this was not his fault, that this is not punishment for anything he did that was bad.

The decrease in energy and the lack of investment in the environment becomes progressive. The contrast between these children and the other children on the ward is great. These children are not interested in what is on the the card with the diagnosis at the end of the bed; it may be available to them, but they do not look at it. They are perfectly willing to be dependent. If we understand what happens in terms of less energy being available, we can see and understand a regression to a state of dependency. As there gets to be less and less energy available, there gets to be a regression to an even earlier state of normal personality development in which there are no relationships at all. The child may be for the most part unrelated, and resent being interfered with. The final step in that direction is coma and, eventually, death.

Returning to practical problems, the decisions we have to make include such questions as should there be treatment at all, or how long should treatment go on. I do not think these decisions should be left too much to the family. We can see the anxiety created by their having to make such decisions. If they are not the right decisions, then what? We, as doctors, can take the blame if there has not been a specific type of treatment brought into the picture. We will get lots of blame as we go along in practice, and some of it, I suppose, is justified. However, the vast majority of it is not, and this we have to learn how to handle. If we have anxiety, our anxiety gets to be reflected in the way we handle both the parents and the child. Therefore, we have to take a look at ourselves in this process. We have to be aware of our own feelings and try not to suppress them, but instead see them for what they are and try to handle them realistically.

Congenital Glaucoma

FRANK D. COSTENBADER, M.D.*
MARVIN L. KWITKO, M.D.†

Glaucoma is that state of increased intraocular pressure which the eye cannot stand without resulting damage. There are certain diurnal variations in intraocular pressure which are entirely within normal range, as well as certain elevations in intraocular pressure due to excitement and physical exertion. In primary glaucoma, the pressure is due to an inadequacy of the aqueous outflow mechanism, or, more rarely, due to an excessive formation of aqueous. Secondary glaucoma may be due to intraocular inflammation, tumors, interference with the vascular circulation, retrolental fibroplasia, congenital anomalies, and a host of other causes.

We would limit our discussion to congenital glaucoma with major emphasis on the primary type. Congenital glaucoma, while not frequently seen (about 1:8000 clinic admissions), is extremely serious, causing from 2 to 13 per cent of all childhood blindness. The condition is bilateral in 70 per cent, and approximately 70 per cent of all cases occur in males.

Some review of the physiology and pathogenesis seems pertinent. Diagnosis, differential diagnosis, and clinical course, especially from the point of view of the pediatrician, will be considered. The treatment and prognosis will be described. Special emphasis will be placed upon distinguishing between congenital glaucoma and adult glaucoma, two entirely different clinical entities.

The aqueous humor (similar in composition to tears), forms in the ciliary body at a fairly regular rate, flows around the lens and out through the pupil, circulating in the anterior chamber, and then into the filtration angle formed by the anterior face of the iris and the posterior surface of the cornea (figs. 1 and 2). There it normally passes through the sievelike trabeculum into the Canal of Schlemm, and, by means of the aqueous veins, is carried to the general circulation. If the aqueous forms at too fast a rate, or if the outflow mechanism is defective, an increasing amount of aqueous is retained within the eye and the pressure goes up, much as in an inflated basket ball.

In the adult (whose sclera is inelastic), this increase in pressure, if not extreme, may cause a minimum of symptoms. However, the compression of the nerve fibers running into the optic nerve causes subsequent optic cupping, typical loss of visual field, and, at a late stage of the disease, irreparable loss of peripheral vision (figs. 3 and 4)

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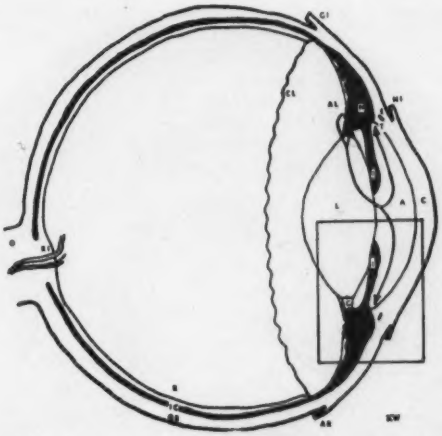

FIG. 1. Sagittal section through the eye showing circulation of the aqueous humor. GI, Medial rectus; N, Ciliary body; E, Schlemm's canal; T, Trabeculum; NI, Conjunctiva; I, Iris; C, Cornea; A, Anterior chamber; L, Lens; G, Zonules; IC, Choroid; OR, Sclera; RI, Retinal artery and vein; O, Optic nerve.

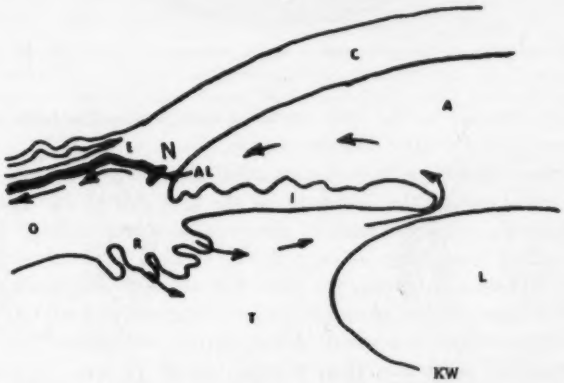

FIG. 2. The filtration angle. O, Sclera; R, Ciliary body; T, Posterior chamber; L, Lens; I, Iris; A, Anterior chamber; AL, Filtration angle; C, Cornea; N, Canal of Schlemm; E, Emissary veins.

On the other hand, the infantile eye whose sclera is elastic shows a gradual expansion of the globe when pressure is elevated; ruptures occur subsequently in Descemet's membrane with resulting hazy cornea and photophobia. Cupping of the optic nerve is a late sign, since distensibility of the

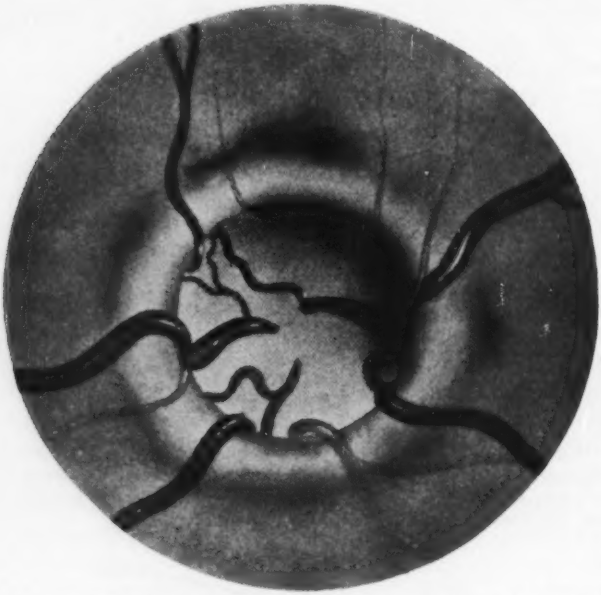

FIG. 3. Excavation of the optic disc in adult glaucoma as seen with the ophthalmoscope.

globe delays damage to the optic nerve. Visual loss is due both to corneal scarring and to nerve fiber damage (fig. 5).

The typical clinical picture of congenital glaucoma is that of an infant seen any time between the first day and the first year of life (rarely thereafter), whose parents state that, 1) the eyes have looked large (and often quite beautiful) since early infancy, 2) the corneas have taken on a blue-gray hazy appearance within the past few days to few weeks, 3) since noticing the hazy cornea, photophobia has been marked, and 4) epiphora, a very evident sign, is present. Examination will then show that the corneal diameter is greater than normal (about 11 mm., below the age of six months), and the corneas have an almost homogeneous blue-gray haze with a somewhat roughened appearance of the surface epithelium. Ruptures in Descemet's membrane can often be demonstrated, and when the anterior chamber can be visualized, it usually is deeper than normal; often the pupil is somewhat dilated. Frequently, the arcade of vessels in the iris is very prominent and can be seen grossly. If the course of the disease is allowed to go on without successful treatment, actual scarring of the previously transparent cornea takes place, and an irreversible haze results. The ciliary body and iris become atrophic, and the Canal

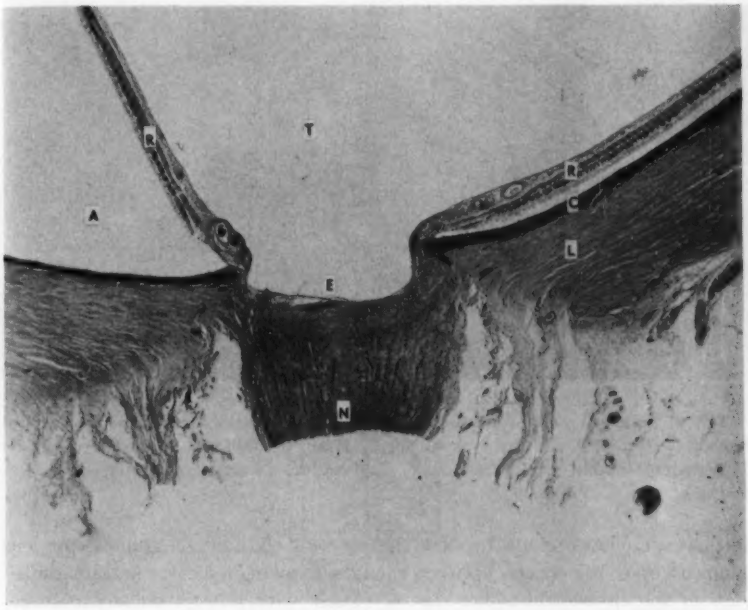

FIG. 4. Excavation of optic disc (microscopic cross section). T, Vitreous humor; E, Excavated optic disc; N, Optic nerve; R, Retina; C, Choroid; L, Sclera; A, Artifactitious separation of retina. AFIP Acc. 787296.

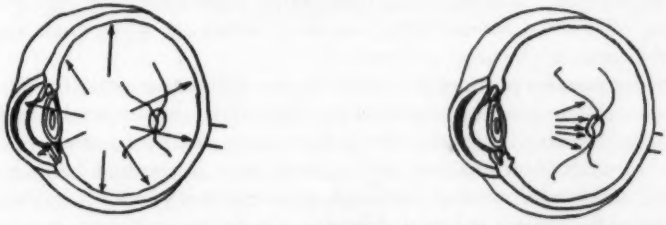

FIG. 5. Tangential section of the eyeball. The elastic infantile eye (left) expands in all directions when the intraocular pressure rises. Much later in the course of the disease, the optic nerve becomes cupped. The inelastic adult eye (right) expands only at its points of weakness, namely the site of the entrance of the optic nerve. There will also be present an associated nasal displacement of the retinal blood vessels.

of Schlemm is compressed and becomes atrophied; the optic nerve becomes cupped as a late sign.

To further confirm the diagnosis of congenital glaucoma, the infant should be placed under a general anesthesia at a deep surgical level. If the

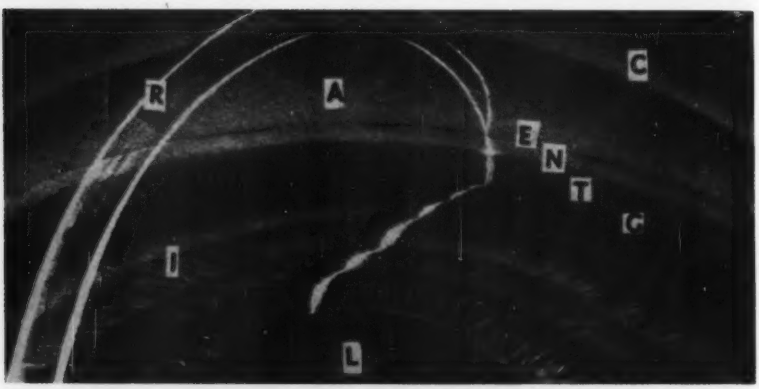

FIG. 6. Enlarged view of the filtration angle as seen with the gonioscope beam of light. C, Cornea; A, Anterior chamber; R, Beam of light; E, Schwalbe's line; N, Trabeculum (nonfiltering portion); T, Trabeculum (filtering portion); G, Ciliary body; I, Iris; L, Pupil.

corneas are clear enough to view the anterior chamber, a gonioscopic lens is placed upon the cornea between the lids, allowing a clearer and magnified view of the anterior chamber (fig. 6). Then, with a hand binocular microscope or a loupe and a bright beam of light, the filtration angle can be viewed. In congenital glaucoma, this frequently shows that the root of the iris is inserted further forward on the cornea than is normal, thus occluding the trabeculum; alternately, a thin glassy membrane may be present, sealing off the interstices. Often, no gross pathology of the angle can be noted in spite of the glaucoma present.

The intraocular pressure is measured by using a tonometer (fig. 7). The foot plate of this instrument is rested on the cornea, and a central plunger of known and graduated weight indents the cornea, moving an arm across the scale. In adult glaucoma, tensions up to 25 mm. of mercury may be considered within normal range, although somewhat suspicious of glaucoma at the upper limits. In congenital glaucoma, the upper limits are not too well defined, but an intraocular tension of above 25 mm. of mercury suggests the possibility of glaucoma, to be confirmed by the other signs and symptoms. A recent study done in this hospital demonstrated that on children known *not* to have glaucoma, the tension varied considerably, depending on the depth of anesthesia, the accurate placing of the tonometer on the cornea, and presumably upon individual variations.

The diagnosis of congenital glaucoma, therefore, is not dependent on one finding, but several. If, for example, the tension would seem to be

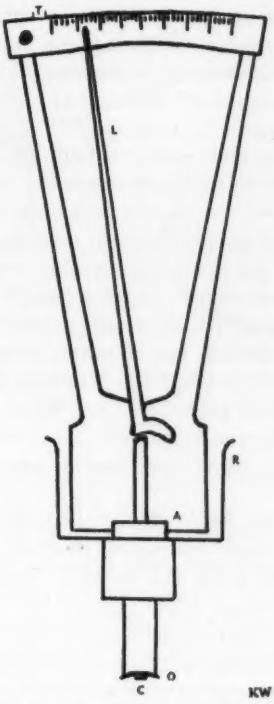

FIG. 7. Shiotz tonometer. T, Scale; L, Lever arm; R, Finger rest; A, weight; O, Foot plate; C, Central plunger.

within normal limits but the patient continues to have photophobia and epiphora, glaucoma probably still exists and damage is taking place. On the other hand, if the tension is elevated somewhat, the cornea crystal clear, and no photophobia or epiphora exists, the presence of glaucoma can be questioned, although the patient must be carefully followed.

The symptoms and signs of congenital glaucoma must be distinguished from 1) corneal abrasion which causes profuse tearing and photophobia, 2) foreign body on the cornea or under the lid also causing epiphora and photophobia, 3) interstitial keratitis (fortunately quite rare now) which causes a hazy cornea with marked epiphora and photophobia, 4) megalocornea, a congenitally large cornea, clear and with no increase in tension, and 5) congenital haze of the cornea, in which the haze is not accompanied by increased tension or other symptoms except decreased visual acuity.

The diagnosis of glaucoma having been suspected by the pediatrician or

the general practitioner is confirmed by the ophthalmologist; thereafter speed in effective treatment is of the utmost importance. A few days of elevated tension may do irreparable damage and make treatment much more difficult. Any unexplained haziness of the cornea or photophobia should immediately suggest the possibility of congenital glaucoma.

While the treatment of adult glaucoma falls into three categories (topical, systemic, and surgical), the adequate treatment of congenital glaucoma is almost exclusively surgical, with some help from the other two categories.

If the globe is not too grossly enlarged (corneal diameter of 14 mm. or greater), or the cornea not too extremely hazy and scarred, the operation of choice is that of goniotomy, which attempts to open the occluded filtration angle. This operation, first suggested by de Vincentiis over 50 years ago, has been, within the last 25 years, repopularized and refined by Barkan, and has greatly improved the prognosis for successful treatment. Before the recent advent of goniotomy, over 90 per cent of all patients with congenital glaucoma eventually became blind, whereas now better than three fourths maintain good and useful vision following successful goniotomy.

In the operation of goniotomy (fig. 8), a special goniotomy knife with a thin blade enters the cornea just inside the limbus, is carried across the anterior chamber to just enter the filtration angle on the opposite side, and is swept in the filtration angle through about one quadrant. If successful, the iris root falls back or the membrane is incised, and the aqueous may then filter into the trabeculum and out through the Canal of Schlemm. Often, more than one such operation is necessary, at times as many as four or five, before successful filtration takes place.

If the globe has been grossly enlarged and the cornea markedly hazy, an external filtering operation would seem to be the one of choice. The iridencleisis (fig. 9), in which an incision is made into the anterior chamber under a conjunctival flap and a pillar of the iris incarcerated in the incision, offers some hope of success and stabilization of tension. At times, the trephine operation, in which a hole is bored in the corneal-scleral junction

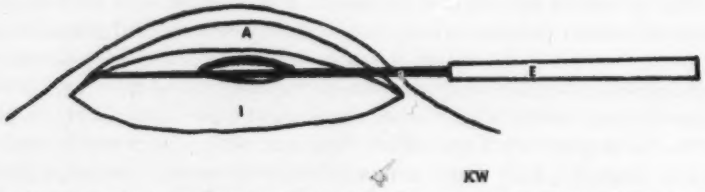

FIG. 8. The goniotomy knife is inserted into the cornea at the limbus and incises the filtration angle. A, Anterior chamber; I, Iris; E, Goniotomy knife.

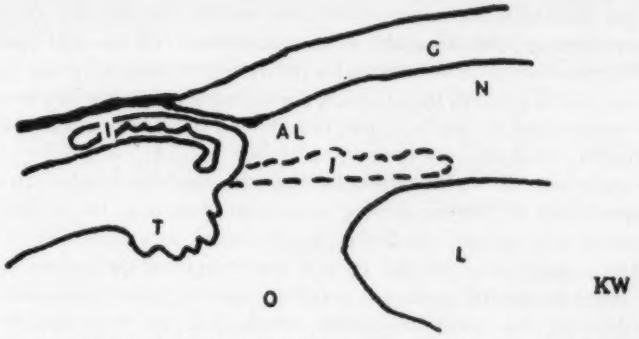

FIG. 9. Iridencleisis. A portion of the iris is incarcerated through the perforated sclera and into the subconjunctival tissue in order to serve as a drainage wick. I, Iris; AL, Filtration angle; T, Ciliary body; C, Cornea; N, Anterior chamber; O, Posterior chamber; L, Lens.

under a conjunctival flap, is done. Unfortunately, the external filtering operations are not often successful, possibly because they are used as late procedures; however, even in Europe, where they are used early in the treatment, they are less successful than goniotomy.

Occasionally cyclodiathermy, a procedure in which diathermy over the ciliary body is performed, is done. This causes some low grade destruction of the ciliary body with decreased formation of aqueous and consequent lowering of tension.

To supplement the treatment between surgical sessions, Diamox, 50 mg. every 6 hours, to suppress the formation of aqueous, and miotics to reduce the size of the pupil and to pull the iris root out of the angle, if possible, are used.

The prognosis in the treatment of congenital glaucoma is directly in proportion to two factors: the age at onset, and the promptness of treatment. The earlier in life the glaucoma is evident, the poorer the prognosis, irrespective of the form of treatment. Thus, those patients in the obstetrical nursery in whom the cornea is found to be hazy respond very poorly to any form of therapy; the result is often a blind, painful eye. We can recall several patients on whom as many as twelve to fifteen operations have been performed with a resultant minimum of residual vision, the tension often still somewhat elevated, and the eyes obviously mutilated. On the other hand, those patients in whom symptoms were not evident until the fourth, fifth or sixth month have a much better prognosis. The late onset suggests that the imbalance between aqueous formation and aqueous outflow is minimal, and thus the restoring of balance by proper treatment is much easier. Even

when the glaucoma is thought to be under control, the intraocular tension must be taken at intervals under general anesthesia and the eyes examined carefully periodically to be sure that a recurrence or residual of the original defect is not still present. In general, if the intraocular tension has been kept under control and the patient symptom-free for six months, recurrence of symptoms is most unlikely.

In conclusion, we would emphasize that the ophthalmologist rarely has the opportunity of first recognizing congenital glaucoma; the pediatrician, the general practitioner, the public health nurse, and others more often have this opportunity. Second, even if the symptoms do not seem to be acute, when congenital glaucoma is present certain appreciable damage is being done to the visual mechanism which may not be reversible even though the glaucoma is finally brought under control. Third, infantile glaucoma differs significantly from adult glaucoma in that adult glaucoma occurs within a nonexpansile globe and causes damage early to the seeing mechanism. In infantile glaucoma the globe is expansile; the increased pressure enlarges the eye, while damaging the iris, retina, and, finally, the optic nerve.

APPENDIX

Twenty-one cases of congenital glaucoma seen by the authors, and listed in order of age of onset. Several of the patients were seen in consultation so that information is not complete. G M C—Good, Maintained, Central; N L P—No light perception; N-Normal; OD-Right eye; OS-Left eye.

Patient	Age of Onset	Eye Examined	Initial Symptoms					Surgical Procedure		Final Result	
			Photophobia	Hazy Cornea	Tearing	Enlarged Eye	Tension mm. Hg	Goniotomy with or without a Goniopuncture. No. Times Performed	Other Procedures Iridencleisis Cyclodialysis Cyclodiathermy	Vision	Tension (mm. Hg.)
1. J K	birth	OD		X		X	50	X			
		OS		X		X	50	X			
2. C P	1 m.	OD		X		X	35	XXX			26
	birth	OS		X		X	55	XXX	X	?	36
3. W R	birth	OD	X	X			?	X			
		OS	X	X			45	X			
4. C H	1 m.	OD	X	X	X		40	X		GMC	20
		OS	X	X	X		40	X		GMC	20
5. E C	3 m.	OD					25			20/15	26
		OS		X		X	40	X		20/200	26
6. V O	3 m.	OD		X		X	50	X		20/200	23
		OS					35			20/20	26
7. M D	3 m.	OD	X	X		X	28		X	10/200	28
		OS					24			20/15	24
8. D W	4 m.	OD	X				50	XXXX			28
		OS	X				50	XXX			
9. D P	4 m.	OD	X	X	X	X	50	X		20/70	18
		OS					20			20/30	24
10. S K	4 m.	OD		X		X	25	X		GMC	N
		OS				X	30	X		GMC	N
11. T M	4 m.	OD	X	X			45	XX		20/20	
		OS	X	X			45	XX	X	NLP	
12. P R	4 m.	OD	X	X			40		X		23
		OS	X	X			40		XX		47
13. S B	5 m.	OD		X		X	48	XX	XX	20/50	N
		OS					30			20/20	N
14. M G	6 m.	OD					18			20/20	18
		OS	X				30	X	XX	20/20	18
15. J L	6 m.	OD				X	50	X		20/30	20
		OS					30			20/20	23
16. T W	6 m.	OD	X	X	X			XX		NLP	24
		OS	X	X	X				X	NLP	15
17. D S	8 m.	OD		X	X		27				23
		OS		X	X		45	XX	XX	(enucleated)	
18. D A	9 m.	OD	X	X		X	40	X		20/20	18
		OS				X	40	X		20/20	11
19. K H	1 y.	OD					25				
		OS				X	35	XX			
20. R B	3 y.	OD		X		X	45	XX		20/30	24
		OS					20			20/20	20
21. W W	9 y.	OD				X	30		X	20/40	35
		OS					18			20/30	30

Chronic Lead Intoxication: A Report of 24 Treated Cases at Children's Hospital

NICHOLAS P. HARITOS, M.D.*

This paper presents a review of 24 cases of chronic lead intoxication treated for the first time at Children's Hospital in the 30 month period extending from January 1, 1957 to June 30, 1959. Cohen and Ahrens[1] have reported a similar group for the preceding seven year period which will be compared with the present group.

The criteria used in defining lead intoxication in this report differ from those used by Cohen and Ahrens, who used broader limitations. The diagnosis of lead intoxication in the present series was made if the patient fulfilled one of two of the following criteria: a pretreatment blood level of .05 mg. per 100 ml. or greater, or a pretreatment urine lead level of .08 mg. per liter or greater. Using these criteria, 24 cases were abstracted from the hospital files. Several more cases were treated during this period; these, however, did not fulfill the above criteria and were discarded.

The etiology of the plumbism in the cases in the current series was on the basis of pica for lead-containing materials, for which a history was obtained in all cases. There were 14 boys and 10 girls in this series. All but one of these were Negroes, a probable reflection of the fact that the clinic population is mainly Negro. Only one patient was under the care of a private physician.

As in the previous paper the patients were grouped into four clinical classifications; asymptomatic, symptomatic, mild encephalitis and severe encephalitis. The symptomatic group included those patients with gastrointestinal symptoms. Those patients classified as having mild encephalitis exhibited mild central nervous system symptoms, such as irritability, lethargy, or a single seizure. Those included in the group with severe encephalitis presented with repeated seizures, and/or prolonged coma. Table 1 shows the results of this classification. The figure of 41.5 per cent due to the encephalitic type, compares well with the previous findings of Cohen and Ahrens.

Table 2 records the age of the patients at the time of the first admission. From this table it is evident that the largest number of cases (50 per cent) were in the 19 to 24 month old age group, and all but one case were under the age of 3 years. In the previous report the largest number of cases occurred in the years from two to three. It was impossible to determine accurately the age of onset of pica.

The clinical findings on admission are summarized in table 3. Many of

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the patients presented with a variety of complaints and findings. Gastrointestinal symptoms were most common and consisted of vomiting, anorexia, constipation, or abdominal cramps. Nine patients had neurologic complaints including headache, lethargy, irritability, and convulsions. The 4 asymptomatic patients were diagnosed by eliciting a history of pica.

Ten patients had physical findings suggestive of infection, whereas eight had neurologic findings, e.g., lethargy, changes in the deep tendon reflexes, positive Babinski sign, clonus, seizures, or coma. One patient who subsequently died presented with repeated seizures and prolonged coma. Seven patients exhibited pallor as compared to only 6 of 43 patients in the previous series. Normal findings were described in six of the children. As is generally expected, none of the children in this series had "gum lines." The diagnosis of lead intoxication was considered at the time of admission in all but one case; in this child, the diagnosis was considered when the initial peripheral blood smear revealed basophilic stippling of the erythrocytes.

A summary of the laboratory results is included in table 4. Hemograms were performed on all patients. In this hospital, a hemoglobin above 10 Gm. per 100 ml. is considered normal for this age and clinic group. Using this value, 18 patients, or 75 per cent, had abnormally low hemoglobin levels as compared to 86 per cent in the previous study. Of these, eight had marked anemia, i.e., a hemoglobin below 8 Gm. per 100 ml. The peripheral blood smear was consistent with the findings of an iron deficiency anemia.

Approximately 80 per cent of these children had a normal leukocyte count (below 15,000 per cu. mm.). Of the 5 children with a leukocytosis, two had evidence of respiratory infection, two had had numerous convulsions,

TABLE 1

Distribution of 24 Cases of Lead Poisoning by Clinical Classification

	Present Series	Cohen and Ahrens[1]
Asymptomatic	9 (37.5%)	28%
Symptomatic	5 (20.8%)	32%
Mild encephalitis	6 (25.0%)	19%
Severe encephalitis	4 (16.5%)	21%

TABLE 2

Age at Admission

12–18 months	6
19–24 months	12
25–30 months	2
31–36 months	3
Over 36 months	1

TABLE 3

Clinical Findings

Symptoms on Admission		Physical Findings on Admission	
Gastrointestinal	11	Infection	10
Neurological	9	Neurological	8
Infectious	3	Pallor	7
Behavioral	2	Normal	6
Ingestion	2	Gum lines	0
Asymptomatic	4		

TABLE 4

Laboratory Results

Hematology		*Cerebrospinal fluid* (10 cases)	
Hemoglobin (24 cases)		Normal	6
Normal	6	Abnormal	3
Over 10 Gm./100 ml.		Protein increased	2
Abnormal		Cells increased	1
8–10 Gm./100 ml.	10	Sugar increased	3
Under 8 Gm./100 ml.	8	Pressure increased	2
White blood cell count (24 cases)		Traumatic	1
Normal	19	*Fasting blood sugar* (4 cases)	
Abnormal (over 15,000/ cu.mm.)	5	Normal	4
Basophilic stippling of RBC (7 cases)		Elevated	0
First smear	4	*Electroencephalogram* (5 cases)	
Later smears only	3	Normal	2
Eosinophilia (over 5%)	3	Abnormal	
Urine (24 cases)		Focal	1
Normal	18	Diffuse	2
Abnormal	6	*X-rays of long bones* (24 cases)	
Proteinuria	2	Normal	3
Glycosuria	3	Abnormal	21
Pyuria	4		
Hematuria	1		

and the last had been hospitalized following the ingestion of cleaning fluid. Only 7 patients had peripheral smears showing basophilic stippling, and three of these only after repeated smears. This differs greatly from the findings of Cohen and Ahrens in whose series almost 50 per cent of the patients showed stippling. Eosinophilia (above 5 per cent) was found in 3 patients, two of whom had negative stool determinations for ova and parasites. Apparently no further investigation of the eosinophilia was carried out in the third case.

Routine urinalyses were performed on all patients. These were all random specimens. The six abnormal urines are summarized in table 4.

Lumbar punctures were performed on 10 patients. Values considered normal for the cerebrospinal fluid were: up to 10 leukocytes per cu. mm., up to 45 mg. per 100 ml. of protein, and up to 60 mg. per 100 ml. for sugar. The 3 patients with abnormal findings had been classified as encephalitic. An abnormally high sugar determination was the most common finding. One lumbar puncture was traumatic.

Fasting blood sugars were determined in 4 patients, and all were found to be within normal limits.

Electroencephalograms were performed on 5 patients, and three were found to be abnormal. Two of these were diffusely abnormal, and one showed a bilateral frontal focus.

X-rays of the long bones were done in all instances. Twenty-one children exhibited the typical increased density of the metaphyses. One case showed increased density of the cranial bones.

Table 5 contains the blood and urine lead levels before and after treat-

TABLE 5

Effect of Treatment on Blood and Urine Levels of Lead

	Blood Lead Level (mg./100 ml. blood)							
	Before				After			
	<.05	.05–.08	>.08	Total	<.05	.05–.08	>.08	Total
Asymptomatic	3	6	0	9	8	1	0	9
Symptomatic	2	2	1	5	2	1	1	4
Mild encephalitis	2	1	3	6	4	2	0	6
Severe encephalitis	2	1	1*	4	1	1	1*	3
Totals	9	10	5	24†	15	5	2	22†

	Urine Lead Level (mg./liter of urine)							
	Before				After			
	<.08	.08–.15	>.15	Total	<.08	.08–.15	>.15	Total
Asymptomatic	2	0	6	8	3	1	3	7
Symptomatic	1	2	1	4	4	0	1	5
Mild encephalitis	3	1	1	5	3	0	1	4
Severe encephalitis	0	1	3	4	1	0	0	1
Totals	6	4	11	21†	11	1	5	17†

* Expired

† Difference in totals due to laboratory mishaps, misplaced samples, etc.

ment. Treatment consisted of the administration of calcium disodium ethylenediaminetetracetate (EDTA) in an amount of 30 mg. per Kg. of body weight per 24 hours dissolved in a 2½ per cent dextrose in half strength Ringer's solution. This was given by hypodermoclysis every eight hours for two 5 day periods with an intervening 3 day "rest" period.

Before treatment was instituted, 15 of 24 patients had blood lead levels of .05 mg. per 100 ml. or greater. After treatment, only 7 of the 21 determinations were in the toxic range; however, the blood level was significantly decreased in almost all cases. The results of treatment on the urine lead levels showed the same decrease. Unfortunately some of the samples obtained were returned from the laboratory labeled as "lost" or "broken in laboratory mishap." By comparing the lead levels and the clinical classification, it is evident that there is no real correlation.

In table 6 an attempt is made to show the results of follow-up examinations. Four patients were not seen for as long as six months after treatment and were considered as no follow-up.

Although the previous series reported three deaths out of 43 cases, only one death occurred in this series. This was a child with prolonged coma, repeated seizures, and markedly elevated intracranial pressure. A craniotomy was performed in an effort to decrease the pressure, but the patient expired after a prolonged downhill course.

Pica ceased in 10 of 19 patients following the first course of treatment. There were residual findings (staggering, continued seizures, behavior problems, and epigastric pain) in only 4 patients, a great improvement over the 45 per cent incidence of residuals in the previous report. Six of the patients were subsequently readmitted for treatment. Of these, three were readmitted on one and three on two occasions. Five of the six patients who

TABLE 6

Follow-up (Six Months After Treatment or Longer)

	Asymptomatic	Symptomatic	Mild Encephalitis	Severe Encephalitis	Total
Deaths	0	0	0	1	1
No follow-up	0	2	1	1	4
Follow-up	9	3	5	3	20
Pica					
Stopped	5	2	3	0	10
Continued	4	1	2	2	9
Residual					
None	8	3	3	1	15
Mild	1	0	2	0	3
Severe	0	0	0	1	1

were readmitted were then cleared of both symptoms and pica. The one patient in whom both symptoms and pica continued had been classified as having severe encephalitis. She continued to have seizures, and required analeptics for their control. It is felt that a high index of suspicion and early vigorous treatment have improved the residual morbidity statistics at this hospital.

SUMMARY

The clinical and laboratory findings in 24 cases of chronic lead intoxication are reviewed. Diagnosis was confirmed in every case by the finding of an abnormal blood and/or 24 hour urine level; there was no correlation between symptoms and these levels. Anemia and "lead lines" in the metaphyseal regions of the long bones as visualized by x-ray were the most consistent findings. Basophilic stippling of the erythrocytes was not a common finding.

Treatment in all cases was with EDTA; six patients required repeat courses of treatment. Only 1 patient is known to have any neurologic residual, which consists of repeated seizures.

The author wishes to extend his heartfelt gratitude to his colleague, Dr. Wellington Hung, whose aid in abstracting a myriad of charts was invaluable in the compilation of this report.

REFERENCE

1. Cohen, G. J., and Ahrens, W. E., Chronic Lead Poisoning, J. Ped. **54:** 271, 1959.

The Editor's Column

Pediatric Ophthalmology Needs a Children's Hospital

The inspiration for these remarks came from Dr. William S. Anderson's "What is the Future of Pediatrics?: A Credo" which appeared in Clinical Proceedings of Children's Hospital (**16:** 346, 1960). I was distressed to learn some dissensions exist within the fields of pediatrics, internal medicine, and general practice over the future of pediatrics as it is now constituted. Apparently there are some proponents who advocate the demotion of pediatrics to the level of one of the subdivisions of internal medicine while others urge it to be synthesized into the boundless field of general practice. This defeatist attitude further implies that the need for a children's hospital will eventually cease.

My distress over this issue is twofold. First, it was my impression that pediatrics had long been established as a needed worthwhile medical specialty and I am surprised to find some members within the specialty itself have challenged that position. Although problems confronting pediatrics have changed with time and recent advances, I would think this change would allow a greater percentage of the total energy to be expended on the prevention and early detection of medical and behavioral problems in children. Using my specialty as a comparison, it is evident that ophthalmology is vigorously moving in the direct opposite to abdication. Through its more energetic coverage of all phases of eye care a multipronged subdivision of the specialty is developing, each subdivision, however, remaining firmly attached to the central core of general ophthalmology. It was significant that Dr. Anderson encouraged furtherance of the development of subspecialties by pediatricians as a partial solution to their worry about the future of pediatrics.

My second distress is over the implication that an advanced hospital designed for special needs of children would cease to be needed in the future. Surely this is not the conviction of those physicians who train other pediatricians.

Although no one in the field of ophthalmology entertains a thought that it will fade away like an old soldier, there are some who do not look upon its subdivision as an advance. The conviction is strong in the minds of some general ophthalmologists that they can adequately perform within all facets of this specialty. However, the fact that subdivision is occurring within their specialty refutes this concept. The subdivision is inevitable since a single mind finds it impossible to master thoroughly and put into practical use the multitude of established basic science facts, the current mountain of research data, and the ever expanding volume of advances in intricate clinical techniques. Moreover, since progress is measured by accumulation of more knowledge and application of this increased knowledge, researchers and teachers tend to focus on increasingly limited areas of the over-all frontier of ophthalmology. Therefore, inherent within the very process which affords pursuit of elegance for ophthalmology are the seeds which eventually flower into the bouquet of its subdivisions. The particular subdivision concerned in this essay is pediatric ophthalmology.

Children's Hospital was the first place in this nation to attract the full time devoted attention of an ophthalmologist. Approximately 20 years ago Dr. Frank D. Costenbader divorced himself from general ophthalmology and funneled his energies exclusively into this subdivision. True, this was to be an inevitable evolution within ophthalmology; it was, however, more than an accident that the birth of this subdivision occurred in this city. The

fact that it was born here and flourished to where it now is a strong and vital part of ophthalmology practiced in this area is testimony in general to the commanding position of pediatrics in this city and in particular to the stimulating atmosphere for pediatric development this hospital has generated during the past half century. This has occurred in specialties other than ophthalmology within this hospital, such as psychiatry, anesthesiology, allergy and roentgenology, but I am not conversant with the relationship between their local and national development. Would all this have come about had there not been a central strong hospital in this community operated solely for the need of children?

The justification for pediatric ophthalmology is the fulfillment of rendering high quality children's eye care associated with a low quantity of disturbing emotional side effects. This can best be achieved by the physician who devotes himself to children's eye problems, who orientates office and outpatient clinic toward children, and who has available good pediatric hospitalization and support by colleagues in other specialties as interested as he in providing superior treatment for children (for example, the pediatric anesthesiologist).

Training in pediatric ophthalmology locally takes place on two levels. The first level occurs during the 3 year residency in general ophthalmology. Each ophthalmology resident at the Washington Hospital Center rotates through a 4 month service at Children's Hospital during the second year of his residency. This level of training brings attention to the future ophthalmologist that children's eye care requires special knowledge of eye conditions peculiar to children and special techniques for working with these little patients. The second level of training is designed for the ophthalmologist who has completed his general eye residency and wishes to develop his skills for doing exclusively pediatric ophthalmology. The outpatient eye clinic and inpatient eye service, as well as associated ophthalmological conditions seen in patients on the general pediatric wards provide the axis about which the major portion of this training in pediatric ophthalmology rotates. This means our Children's Hospital is vital to the continuance of training in this specialty. I suspect this same dependence for specialty training exists in other fields, such as psychiatry and anesthesiology.

Having been inspired by Dr. Anderson's dedicated beliefs as to the future of pediatrics and children's hospitals, I wish to state my credo of pediatric ophthalmology:

> I believe the emphasis on children's eye problems was sadly lacking in the past, is currently receiving proper attention in a few isolated areas of our nation, and will eventually grow to be one of the major subdivisions of general ophthalmology throughout the nation.

I believe pediatric ophthalmology is dependent upon pediatricians being alert to eye problems, detecting them at a young age, and instructing parents to seek early care.

I believe that a children's hospital offers the best setting for ophthalmological care of children and training of those who wish to practice pediatric ophthalmology.

MARSHALL M. PARKS, M.D.

Book Reviews

Synopsis of Ophthalmology. By WILLIAM H. HAVENER, M.D., 288 pages, 189 illustrations, St. Louis: The C. V. Mosby Company, 1959, $6.75.

This is a concise presentation of visual and eye problems that is helpful for the medical student and a handy reference for any physician, particularly the general practitioner. The illustrations are excellent, plentiful, and considerably enhance the text.

This review was conducted from the point of view of a pediatric ophthalmologist evaluating the book for the pediatrician. Since the book was not written specifically to fulfill the needs of the pediatrician, any criticism concerning a deficiency in satisfying this limited purpose is therefore unfair to the author.

The pediatrician will find that his difficulty in using this text as a quick reference is also shared by the specialist in geriatrics. Each will have to thumb over and weed out a large amount of material that is irrelevant to his practice. In developing a synopsis for over-all use by all physicians this lack of emphasis of ophthalmological material which would benefit a certain specialist in his limited area of practice is inevitable.

The pediatrician would not be helped if he sought the answer as to how amblyopia in one eye can exist in the absence of strabismus, for there is no discussion of anisometropia. Yet, percentage-wise, anisometropia accounts for as much amblyopia as strabismus. The section dealing with strabismus needs to be expanded for the pediatrician since there is no reference to divergent strabismus, nor any enlightenment about the use of a bifocal correction or parasympathicomimetic drugs in accommodative strabismus. The chapter on developmental anomalies of the eyes is too brief to be of much value to the pediatrician; however, the discussion of nasolacrimal duct impatency is good. Only nine lines on congenital glaucoma and only five diseases discussed in this section leaves much

unsaid. Nor will the pediatrician be satisfied with the meager comments about the rubella syndrome, congenital toxoplasmosis, and retrolental fibroplasia. Also he will find nothing on Marfan's syndrome. The reviewer could not find the word nystagmus in the book although there is a chapter on neuro-ophthalmology. Ptosis is defined in two different places in the text, but not discussed.

The format is excellent; the book is well edited and adequately indexed. There are many fine little sidelights about this book, and one of the gems is the open discussion of what is an ophthalmologist, oculist, optician, orthoptist, and optometrist. From a strictly ophthalmological point of view, this isolated section makes the book worthwhile.

MARSHALL M. PARKS, M.D.

Premature Babies; Their Nursing Care and Management. By A. K. GEDDES, M.D., 215 pages, 13 figures, Philadelphia: The W. B. Saunders Company, 1960, $4.50.

This book was written primarily for the guidance of the nurse caring for the premature infant and is directed toward the nursing care of the premature infant in the hospital. Starting with the infant in the delivery room, the care of the infant is discussed in all aspects. Complications which may arise, drug therapy, feeding, abnormalities which must be noted, and some nursing techniques are dealt with in short, clear, concise chapters.

The use of oxygen in a premature nursery and retrolental fibroplasia are fully discussed. The physical plant of a premature nursery with respect to size, isolation, visability, supplies and equipment needed, and examining rooms are new chapters which give this book particular value. Emphasis is given to the need in a well functioning premature nursery for a skilled pediatric nurse well versed in the care of the premature infant and possessing the ability to educate a good staff. Further points receiving emphasis are an efficient and well publicized transport service, better prenatal care and research, and social and medical follow-up.

Even though there is a certain amount of oversimplification of some of the problems, the short summaries found at the end of each chapter are helpful in emphasizing the most important factors in the nursing care and management of premature infants.

MARY Z. SHERWOOD, R.N.

The Kinetics of Cellular Proliferation. Edited by FREDERICK STOHLMAN, JR., M.D., 456 pages, illustrated, New York: Grune and Stratton, 1959, $4.75.

This book represents the proceedings of a conference on the Fundamental Problems and Technics for the Study of Kinetics of Cellular Proliferation

held in Salt Lake City, Utah, January 1958. Although the conference was concerned with the cell systems in general the major focus was on hematopoesis and lymphopoesis. Participation in the meeting was limited to those individuals who have or are presently active in the field of cellular proliferation. The first six sections of this volume consist of presentation and discussion of the classical morphologic, biochemical and radioisotopic methods, results and interpretation of erythrocyte, leukocyte, and platelet production and survival and their regulatory factors. The last section presents mathematical consideration pertaining to the topic.

The material in this volume is well presented and edited and covers the topic in a complete manner. Although it will not be of much interest to the practicing physician it will be of considerable value to the hematologist and anyone interested in the kinetic aspects of cellular proliferation.

SANFORD LEIKIN, M.D.

The Surgeon and the Child. By WILLIS J. POTTS, 225 pages, illustrated, Philadelphia: W. B. Saunders Company, 1959, $7.50.

This is a book which thrilled the reviewer with astute observation, homey discussions, and a splendid philosophical approach to the field of pediatric surgery. On the whole, however, it was disappointing because while it promised much, it unfortunately did not fulfill that promise.

On the positive side, the first three chapters which have the title of "The Cry of Children," "The Deformed Child," and "The Heart of the Child" should be required reading for all those who aspire to perform surgery upon a pediatric patient. These chapters express a philosophy which is indeed splendid. They show an obvious depth of understanding on the part of the writer for the child and the child's individual, intimate day by day problems. All too often these are disregarded by the parent and the surgeon alike.

On the negative side, it is disappointing that a man of the vast experience and academic background of Dr. Potts should write a text which produces so few statistical results. Diagnosis and treatment are discussed in a manner which is often superficial. There is no bibliography. There is no consistent form used throughout each chapter. In one section separate headings may be used for such things as diagnosis, symptoms, or signs, and in the following section they are not so separated. The illustrations should be greatly increased. The general feeling of the reviewer is that as though a number of essays were well written separately and at individual times and then placed together in book form.

GEORGE WILLIAM WARE, M.D.

Pediatric Nursing. By GLADYS S. BENZ, Fourth edition, 572 pages, 109 illustrations, St. Louis: The C. V. Mosby Company, 1960, $6.00.

The book presents a good over-all picture of pediatric nursing. The author has a particularly good approach in dividing the book into sections entitled, "Developing Skills in Child Care." "Growth and Development," "The Newborn," "Diseases Affecting the Body Systems," "Infectious and Communicable Diseases," and "The Child in the Community."

In writing about developing skills she speaks of the importance of "mothering." "The nurse must learn to feel with the child." "She must have a broad knowledge of children and a warm and diffuse communication with them." "Attitude rather than time is often the most important factor."

Many examples are presented describing behavior at different ages and under a variety of circumstances. Workable suggestions are offered as to what one can do to promote better understanding of this behavior and better communications between the child, the parents, and the nurse.

The chapter entitled "The Safety Program and Child Care," discusses the need for a knowledge of growth and development to help avert accidents as well as the need for safety rules and devices.

It is quite helpful to have in this text several topics which have been omitted or too briefly outlined in other pediatric nursing books, i.e., nursing the child in the home, care of the child in the respirator, rehabilitation, mental retardation, and orthopedic conditions relevant to pediatrics; a larger number of pediatric surgical problems are also included.

An extensive and up-to-date reference list is included at the end of each chapter.

Miss Benz writes in such a manner that pediatric nursing becomes a challenging and worthwhile experience.

HELEN N. KIER, R.N.

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